PREVALENCE OF RETINOPATHY OF PREMATURITY IN PREMATURE INFANTS

THIS DISSERTATION IS SUBMITTED TO SANTOSH UNIVERSITY IN PARTIAL FULFILMENT OF THE UNIVERSITY REGULATIONS FOR THE AWARD OF THE DEGREE OF

MASTER OF SURGERY

(OPHTHALMOLOGY)

Submitted By

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(2020-2023)

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INDEX

SERIAL NO.	CONTENTS
1	INTRODUCTION
2	AIM AND OBJECTIVES
3	REVIEW OF LITERATURE
4	MATERIALS & METHOD
5	OBSERVATION AND RESULTS
6	DISCUSSION
7	CONCLUSION
8	SUMMARY
9	BIBLIOGRAPHY
10	ANNEXURES PROFORMA INFORMED CONSENT MASTER CHART

LIST OF TABLES

S.NO.	TABLE	PAGE NO.
1.	GENDER DISTRIBUTION	39
2.	GESTATIONAL AGE AND ROP	40
3.	BIRTH WEIGHT AND ROP	41
4.	SEX DISTRIBUTION AND ROP	42
5.	OXYGEN THERAPY AND ROP	43
6.	APNEA AND ROP	44
7.	VENTILATION AND ROP	45
8.	ANEMIA AND ROP	46
9.	BLOOD AND BLOOD PRODUCT TRANSFUSION AND ROP	47
10.	FREQUENCY DISTRIBUTION OF STAGES OF ROP	48
11.	ZONE DISTRIBUTION OF ROP	49

LIST OF GRAPHS

S.NO.	GRAPH	PAGE NO.
1.	GENDER DISTRIBUTION	39
2.	GESTATIONAL AGE AND ROP	40
3.	BIRTH WEIGHT AND ROP	41
4.	SEX DISTRIBUTION AND ROP	42
5.	OXYGEN THERAPY AND ROP	43
6.	APNEA AND ROP	44
7.	VENTILATION AND ROP	45
8.	ANEMIA AND ROP	46
9.	BLOOD AND BLOOD PRODUCT TRANSFUSION AND ROP	47
10.	ZONE DISTRIBUTION OF ROP	50

LIST OF FIGURES

S. NO.	FIGURES	PAGE NO.
1.	ZONES BORDERS AND CLOCK HOURS	27
2.	IMMATURE RETINAL VASCULARIZATION	28
3.	STAGE 1 OF ROP	28
4.	STAGE 2 OF ROP	29
5.	STAGE 3 OF ROP	30
6.	STAGE 4A AND B OF ROP	31
7.	STAGE 5 OF ROP	31
8.	PRE PLUS DISEASE	32
9.	PLUS DISEASE	32
10.	AGGRESSIVE POSTERIOR ROP	33
11.	SPECULUM INSERTION	36
12.	EXAMINATION OF ROP	37

LIST OF ABBREVIATIONS

APROP – Aggressive posterior retinopathy of prematurity

BW – Birth weight

CA – Chronological age

ELBW – Extremely low birth weight

GA – Gestational age

HDI – Human development index

IGF – Insulin like growth factor

ICROP – International Classification of retinopathy of prematurity

LBW – Low birth weight

NICU – Neonatal intensive care unit

OPD – Outpatient Department

PCA – Post conceptional age

POG – Period of gestation

ROP - Retinopathy of Prematurity

RLF – Retrolental fibroplasias

RF – Risk factors

TE – Time of examination

VEGF - Vascular endothelial growth factor

INTRODUCTION

Preterm birth can impose challenges on the developing ocular system, resulting in the various visual manifestations of varied significance and pathological scope. This ocular condition can pose severe vision loss.

Retinopathy of prematurity (ROP), or retrolental fibroplasias as it was originally named, had life span as a 20th century disease. Over the previous 59 years this disease was first correlated with prematurity by Terry in 1942, a plethora of literature has emerged on retrolental fibroplasia (RLF) and retinopathy of prematurity (ROP) [1].

It has been responsible for the two largest "epidemics" of blindness in neonates in modern times. These outbreaks of the disease occurred 25 years apart in the mid-1950s and late 1970s approximately [(1)]. It has been an "oxygen epidemic" from the late 1940s through the mid-1950s.

ROP is a bilateral proliferative retinopathy, which occurs in premature infants with low birth weight (LBW) who often have been exposed to higher concentrations of oxygen & effects the developing retinal blood vessels. It emerges out as one of the leading causes of preventable blindness and accounts for up to 10% of childhood blindness in developed countries [2]. Ever since 1940s, with the arrival of improved facilities and treatment, the survival rate of premature infants has been increased & recognized as an important cause of childhood visual impairment and blindness [2].

Its prevalence in moderately developed countries human development index; HDI (30-100) is as high as 60% in premature babies & in poorly developed countries, which includes India, is 3 to 13%. The incidence in highly developed countries, those with a HDI of 1-3% is 3- 13. Its incidence is higher in babies of low gestational age and LBW [3,4]. Major risk factors of ROP include hyperoxia, sepsis, blood transfusions, acidosis, antioxidant deficiency, patent ductus arteriosus, apnea and intraventricular hemorrhage.

A high concentration of oxygen therapy was previously thought to be the prime factor in the development of ROP. However, reports have found ROP without oxygen therapy. Even after oxygen therapy all premature infants does not develop ROP [5]. ROP can be prevented by a simple screening test done within a few weeks after birth [6].

In India, ROP screening should be done in all preterm neonates who are born <34 weeks of gestation and/or <1750 g birth weight; as well as in babies 34-36 weeks of gestation or 1750-2000 g birth weight if they have risk factors for ROP. Screening should start by one month after birth, as early as 2-3 weeks of age in infants born <28 week or <1200g birth weight [7]. The overall extremely low birth weight (<1000gms) infants are three times more likely to have a vision of less than 6/60 than those born at term [8].

Congenital ocular anomalies are also commonly encountered in premature babies. Most congenital anomalies are present long before the time of birth, some in the embryonic period (up to the 7th week of gestation) and other in the fetal period (8th week to term). Various ocular congenital anomalies which are commonly seen are Microphthalmos, Microcornea, iris and choroid coloboma, congenital cataract, and congenital nasal duct obstruction etc.

Various components that affect the early detection and management of ROP are lack of awareness, resources, skilled personnel, financial constraints, and lack of screening protocols. The diagnostic guidelines must take into account the local health-care facilities available for neonatal care.

Retinal examination of infants at risk for ROP using the Ret-Cam digital camera system using wide angle lens with interchangeable high magnification lenses that allows photographic documentation permitting remote interpretation of images and is being used for telemedicine increasingly worldwide [9]. The gold standard modality for its diagnosis is an indirect ophthalmoscope.

The danger for ROP development arises from the fact that a premature baby's retinal development must proceed in a different setting. Even in room air, the infant's retina develops hyperoxia, which results in a reduction in levels of VEGF and for a time, vasculo-genesis is halted between the vascular and avascular retina [10]. Moreover, IGF-1 levels fall from in utero levels after birth, due to IGF-1 loss which is provided by the placenta and the amniotic fluid [11].

The international classification of ROP (ICROP) was invented in 1984 and further expanded in 1987 [12].

ROP is classified at first on the basis of location of the lesion of abnormal vascular development relative to the optic nerve (zone), second on the basis of degree of abnormality (stage), third on the basis of the presence or absence of dilated and tortuous posterior pole vessels (plus disease), fourth on the basis of the extent of disease (clock hours) and finally on retinal sequelae as the disease involutes. Severity of disease involves zone, stage and presence of plus disease.

The treatment should be done at the earliest, preferably within 24 hours and not later than 72 hours. It is recognized on the retinal ablation theory. It targets the avascular region of the retina with the intention of reducing angiogenic growth factor production [13].

The treatment of choice for ROP is peripheral diode laser photocoagulation [13,14]. Although, acute risks of laser photocoagulation include corneal oedema, intraocular hemorrhage and cataract [15]. Post – operatively it is expected that eyelids have moderate oedema and erythema for 3-4 days. However, there may be mild conjunctival congestion, chemosis and subconjunctival hemorrhage for a few days.

In case of excessive manipulation during treatment and especially in eyes with poorly dilating pupils, topical steroids are prescribed 3-4 times a day to reduce any inflammation and specially to decrease the risk of post-laser posterior synechiae formation.

If there is not enough regression or focal areas of active new vessels are seen, secondary treatment is done to laser free areas and around the active proliferation.

The purpose of the study is to know the prevalence of ROP in extreme LBW as it is the most significant problem.

To minimize development of complications such as:

1. Myopia
2. Temporal vitreoretinal fibrosis with dragging of the disc/extensive retrolental fibrovascular tissue.
3. Secondary angle closure glaucoma
4. Total retinal detachment

REVIEW OF LITERATURE

Retinopathy of prematurity (ROP) was defined as a progressive disorder seen exclusively in premature infants of low birth weight, where in a fibrous tissue is formed behind the lens, resulting in blindness and severe visual impairment. This disease was not commonly seen when it was first described, and hence had little interest but it became a major problem to all ophthalmologists and pediatricians about 10 years later. So, there is need to know the prevalence and risk factors of this disease among high-risk babies. Its prevalence in moderately developed countries human development index; HDI (30-100) is as high as 60% in premature babies & in poorly developed countries, which includes India, is 3 to 13%.

Milind Suryawanshi et al in (2022) examined 106 infants who were eligible for ROP screening in which 15 infants developed ROP. Out of the 15 infants with ROP, 7 were male and 8 were female. Prevalence of ROP was 14.15%. The mean gestational age was found to be 31.26 weeks and mean birth weight was recorded 1.348 kg. Amongst 15 ROP cases, 12 (80.00%) were classified into classic, 02 (13.34%) into aggressive posterior retinopathy of prematurity (APROP), and 01 (06.66%) into hybrid. Prevalence of APROP which was 13.34%.[16]

Dr. Bela Shah et al in (2022) found that proportions of ROP increased significantly with an increase in gestational age. Oxygen therapy correlated significantly with the development of ROP. Hyperbilirubinemia requiring phototherapy (80 %), sepsis (73 %), NEC (Necrotizing Enterocolitis) (35 %), HMD (Hyaline Membrane Disease) (41 %), PDA (Patent Ductus Arteriosus) (25 %) and IVH (Intraventricular Hemorrhage) (3.5 %) were found to be statistically significant for ROP development. Laser treatment was found to be very effective in regression of ROP.[17]

Maria Margarita Parra et al in (2022) reviewed 1,691 infants and included 727 (43%) males and 94 (57%) females. Mean gestational age was 32.44 ± 2.6 weeks (range 24-39) and mean birth weight was 1,536 ± 404 grams (range 650-3,700). Mean postmenstrual age at the time of examination was 36.72 weeks (range 26-60). The mean gestational age of preterm infants with ROP was 28.66 ± 2.87 weeks (range 24-38); the mean birth weight was 1,230 ± 538 grams (range 650-2,100). The proportion of ROP in less than 1,000 grams was 42.1%, and of them, 42.6% presented affection in zones I and II. The most frequent risk factors were sepsis and supplementary oxygen and 8.6% of neonates with ROP required surgical treatment.

The global prevalence of ROP was 17.5%.[18]

Thiago Gonçalves Dos Santos Martins et al in (2022) studied 773 patients. The sample consisted of 288 examinations performed by indication of gestational age ≤32 weeks: 118 (42.4%) in 2015, 105 (42.2%) in 2016, 65 (26.4%) cases in 2017. There were 329 evaluations indicated due to birth weight: 113 (40.6%) in 2015, 108 (43.4%) in 2016, and 108 (43.9%) in 2017. The prevalence of associated risk factors was 97 (34.9%) cases in 2015, 96 (38.6%) in 2016, and 54 (22%) in 2017, followed by mechanical ventilation with 82 (29.5%) cases in 2015, 64 (25.7%) in 2016 and 41 (16.7%) in 2017, and continuous positive airway pressure with 59 (21.2%) cases in 2015, 72 (28.9%) in 2016, and 46 (18.7%) in 2017. For the other indications, the evaluations performed due to congenital syphilis were the majority in the 3-year period of the study, with 55 (19.8%) newborns in 2015, 54 (21.7%) in 2016, and 59 (24.0%) in 2017. The most prevalent ophthalmologic diagnosis was retinopathy of prematurity, with 79 (35.3%) cases in 2015, 64 (32.2%) in 2016, and 41 (24.1%) in 2017.[19]

Niusha Kasiri et al in (2022) examined 812 at-risk infants, 807 met the inclusion criteria. ROP was observed in 316 (39.0%) of the 807 premature infants, with types I and II in 142 (45.0%) and 174 (55.0%) infants, respectively. The mean GA (29.6 ± 2.5 weeks versus 30.7 ± 2.5 weeks) and BW (1,203.6 ± 382.8 g versus 1,333.5 ± 386.5 g) were significantly lower in infants with ROP than in those without ROP (both $P < 0.001$). GA ($r = 0.80$; $P < 0.001$) and BW ($r = 0.85$; $P < 0.001$) had a strong positive correlation with ROP, and the correlation increased as GA and BW decreased (all $P < 0.001$). Associated risk factors in infants with ROP, in order of frequency, were oxygen therapy (22.8%), respiratory distress (16.1%), phototherapy (14.6%), blood transfusion (5.7%), apnea (4.1%), mechanical ventilation (1.6%), and intraventricular hemorrhage (0.9%). Multivariate regression analyses showed low GA and BW (both $P < 0.05$) as independent predictors of ROP.[20]

Lemaître D et al in (2021) surveyed 114 children, among whom 32 (28.1%) children received treatment for indications outside the ETROP guidelines for both eyes. The indications outside the guidelines were persistent stage 2 or 3 ROP that showed no evidence of regression after 41 weeks of corrected gestational age (11 children; 34.4%), pre-plus stage (11; 34.4%), difficulties in disease staging (7; 21.9%), type 2 ROP with plus disease (2; 6.2%), and treatment due to logistical difficulties (1; 3.1%; hospitalized in neonatal units hundreds of miles away from our department, with no fundus examination possible in the neonatal unit.[21]

Fekri Y et al in (2021) studied 400 preterm infants (57.2% male and 42.8% female), 107 (26.8%) infants had ROP of whom 23 (21.5%) needed treatment (i.e., 5.8% of all infants need treatment). There were 4 (3.7%), 29 (27.1%), and 74 (69.2%) infants with zone I, II, and III, respectively.

There were 91 (85%), 11 (10.3%), and 5 (4.7%) infants with stage I, II, and III, respectively. Multivariate logistic regression analysis showed that parental consanguinity (P=0.008), gestational age less than 32.5 weeks (P<0.001), and birth weight less than 1725 g (P<0.001) were associated with a 2.3-fold, 4.1-fold, and 4.5-fold increased risk of ROP, respectively. Study showed that one quarter of the premature infants had ROP.[22]

Ibraheem Samih Awad et al in (2021) examined 100 newborn infants, of them 22 cases affected with ROP and 78 cases were free. There were 11 cases of ROP at stage I, 8 cases of ROP at stage II, and 3 cases of ROP at stage III. Prevalence of ROP was 22%.[23]

Dilip Manikantan D et al in (2021) examined 86 participants, 53.5% were male, 50% belonged to gestational age of > 32 weeks, 73.3% were weighted less than or equal to 1500 grams and 26.7% were >1500 grams, 57% participants' mothers had normal delivery, 83.7% of the mother had singleton delivery and 76.7% required oxygen supplementation. Among infants with birth weight less than or equal to 1500 grams, 23.8% had ROP, infants whose mother had gestational age <30 weeks, 33.3% infants who received oxygen supplementation had ROP. Among the study infants who had sepsis 58.3% had ROP. In the present study participants who received exchange transfusion 59.3% had developed ROP, variables like phototherapy, mode of delivery and twin delivery were not statistically significant. 27.9% participants were found to be diagnosed with retinopathy of prematurity.[24]

Q Lumin et al in (2021) conducted a meta-analysis of the prevalence of ROP on 22 separate populations. The prevalence in mainland China was 9.284%. It was negatively correlated with birth weight (BW) and gestational age (GA). 50 independent meta-analyses were observed to be related to environmental exposure factors of ROP. Thirty of the 50 meta-analyses had results that were significant at p values less than 0.05. The first 3 risk factors with the largest combined effect size were GA≤34 w, bronchopulmonary dysplasia (BPD) and BW≤2,000 g.[25]

Yasmim Laila Fragoso Cestari et al in (2021) conducted a gender analysis that revealed small gender-related differences, approximately 52.9% of affected neonates were males and 47.1% females. As to race/color, approximately 72.7% were white, 12% were brown and 2.7% were black. Retinopathy of prematurity affected 32.3%.[26]

Aly H et al in (2020) studied 818,945 neonates. The overall mortality was 16.2% & prevalence of ROP was 17.5%. There was a significant trend for increased ROP over the years. Severe ROP was also significantly increased & was associated with a significant trend for increased median LOS in survived infants. Mortality was significantly decreased whereas NEC & severe NEC did not change over time. The study concluded increased ROP & severe ROP over the 16 years of the study period. This trend was associated with a significant increase in the LOS in survived infants without change in NEC.[27]

Woldemariam EB et al in (2019) conducted a cross-sectional retrospective study on 301 samples of the pediatrics out-patient eye clinic medical records, at Minilik II referral hospital, from March to April 2020 & Data was collected from medical records of all infants visiting the hospital for eye problems from January 2018 to December 2019. The prevalence of ROP among infants in this study showed 39 (13%). Of these, more than half (56.4%) were Zone II + Stage 1, followed by Zone II + stage-2, and Stage-5; 12.8% and 10.2% respectively. Birth weight, oxygen therapy and sepsis were the factors. The prevalence of ROP in the study was (13%).[28]

Zarei M et al in (2019) conducted a retrospective cohort study on infants who had undergone screening for ROP at Farabi Eye Hospital, between March 2016 and March 2017. Data were analyzed based on the presence of extreme prematurity (gestational age ≤28weeks), extremely low-birth-weight (≤1000g), and multiple- gestation (MG) infants. The prevalence of ROP was 27.28% among all screened infants, 74.4% for extremely preterm (EP) infants, 77.5% for extremely low birthweight (ELBW) babies and 27.25% for infants from MG pregnancies. On multivariate analysis, gestational age, birthweight and history of transfusion were found to be significantly associated with ROP. More advanced stages of ROP were observed in EP and ELBW infants. Birth weight, history of transfusion and intubation were not associated with increased risk of ROP in EP infants, while gestational age and history of transfusion were significant risk factors for ROP in ELBW infants. Gestational age and birth weight were significantly associated with ROP in infants from MG pregnancies in multivariate analysis. ROP remains a commonly encountered disease, especially in ELBW and EP infants.

The history of transfusion may have a role in stratifying the risk for ROP and guiding future screening guidelines.[29]

Azami et al (2018) conducted 42 research of 18,000 babies. [30]

Edy Siswanto et al (2017) studied incidence of ROP and possible risk factors associated with the development of ROP in preterm infants in Indonesia & reported incidence of all stages of ROP in infants with a gestational age of<32 weeks ranged from 18–30%. One study showed that ROP also occurred at older gestational ages. Blindness due to ROP was seen in infants up to 35 weeks and with a birth weight of 2000 g.Retinopathy of prematurity is an important cause of ocular morbidity and blindness in Indonesia. The overall incidence of ROP in infants born below 32 weeks in Indonesia is higher than in developed countries, and it is seen in infants with older gestational ages.[31]

Gloria Isaza et al (2015) studied Retrospective case series at a single tertiary care Neonatal Intensive Care Unit. Totally, 14 of 47 (29.8%) infants were classified with zone I ROP and 33 (70.2%) as posterior zone II ROP. The incidence of treatment was higher in the zone I ROP (85.7%) compared to posterior zone II ROP (48.6%). The presentation of infants with ROP in the zone I and posterior zone II are very similar. The clinical course in the zone I ROP is faster and more aggressive than posterior zone II. Due to their atypical morphology and rapid progression, appropriate recognition, and classification of ROP is needed for adequate and timely treatment.[32]

Yousef Alizadeh et al (2015) conducted cross-sectional retrospective study which included all preterm infants with birth weight ≤2500 g and/or gestational age ≤36 weeks. Among 310 infants, ROP was diagnosed in 64 (20.6%) of referred preterm infants, these included stage I in 48%, stage 2 in 29%, and stage 3 or higher disease in 23% of subjects. Mean gestational age (GA) and birth weight (BW) in the ROP–affected infants was 30.18 ± 2.28 weeks and 1,422.8 ±420.8 g, respectively. Low BW, low GA, oxygen therapy, phototherapy, blood transfusion and apnoea were risk factors for ROP.[33]

Ebtisam K Al Alawi et al (2015) examined 1795 premature infants (gestation age ≤32 weeks, birth weight ≤1500 g) admitted to the Neonatal Intensive Care Unit. ROP was detected in 367 (20.4%) infants. There were 68 (18.5%) infants with stage III ROP, 21 infants with Stage III ROP with threshold, and 5 infants with stage IV ROP requiring vitreoretinal surgery. There were 203 (80%) infants with a birth weight <1000g. Birth weight of <1000g was significantly associated to ROP .[34]

Baljeet Maini et al (2014) conducted a prospective, observational cohort study. A total of 148 infants ≤ 34 weeks gestation at birth, completed the study protocol. Over all incidence of ROP (any stage) was 44.6%. Severe ROP was mainly detected in <1200g birth weight and/or <30 weeks gestational age. low birth weight (<1200 g), low gestational age (<30 weeks), acidosis and blood transfusion were associated with babies in severe ROP Low birth weight and low gestational age emerged as independent significant risk factors along with blood transfusion and acidosis. Antenatal betamethasone may be preventive for severe ROP.[35]

AIM AND OBJECTIVES

AIM :

TO STUDY THE PREVALENCE OF RETINOPATHY OF PREMATURITY IN PREMATURE INFANTS.

OBJECTIVES :

1. TO ESTIMATE THE PREVALENCE OF ROP.
2. TO DETERMINE THE RISK FACTORS OF ROP.
3. TO COMPARE THE PREVALENCE OF ROP IN MALE AND FEMALE BABIES.
4. TO COMPARE THE PREVALENCE OF ROP BETWEEN
    - GESTATIONAL AGE 28 WEEKS - 30 WEEKS
    - GESTATIONAL AGE 30 WEEKS -32 WEEKS

MATERIALS AND METHODS

Organization : Department of Ophthalmology & Pediatrics, Santosh medical college and hospital , Ghaziabad.

Study Setting : OPD based

Type of study: Cross sectional study.

Study population: Neonates with Birth weight (BW) <1500 grams & Gestational Age (GA) <32 weeks.

Study Period: August 2020-August 2021

Consent & Approvals: Ethical approval will be taken from the institutional ethical committee.

Sample size: Based on formula

$$n = Z^2_{1-\alpha}/2/e^2$$

where Zα= 1.96 at 5 % level of significance e = 27.73% incidence rate (as per previous study)

n= [(1.96)²/ (.2773)²]

n= 50

50 cases will be taken as sample size[36]

INCLUSION CRITERIA:

1. Premature infants of both sexes.
2. Birth weight less than 1500 grams.
3. Gestational Age less than 32 weeks.

EXCLUSION CRITERIA:

1. Systemic congenital abnormalities.
2. Congenital abnormalities of one or both eyes.
3. Infant hemodynamically unstable.
4. Consent not given by the parents/ guardian.

METHODOLOGY

A fixed time was arranged for screening. Parents of admitted cases were pre informed and consent taken. Proper history was taken. Dilatation was done 15- 20 minutes before examination.

Babies were evaluated in temperature controlled clean environment where risk of hypothermia, infection, and apnea were minimum. For patient in NICU the screening was done in open case system incubation. The child was fed and burped an hour before the examination to minimize the risk of vomiting.

The anterior segment was first evaluated with torchlight and pupillary reactions were noted. The neonates were examined without sedation. Pupils were adequately dilated with 0.5% tropicamide and 2.5% phenylephrine instilled twice, 20 min before evaluation. Excess drops were wiped away each time. Smallest size wire speculum was used to retract the lids. Screening of ROP was done by indirect ophthalmoscopic examination of retina and anterior segment using VOLK +20 D lens.

Babies were comfortably wrapped & placed flat on examination surface. An assistant restrained babies' arms & stabilize the head.

First the anterior segment was evaluated with +20D lens under illumination of Indirect ophthalmoscope for any rubeosis, persistent tunica vasculosa lentis and congenital ocular abnormalities. Staging was performed using international classification.

The disc and posterior pole were evaluated for signs of severe ROP, including plus disease or any stage of ROP / avascularity / vascular tufts and loops in Zone 1. Examination was continued all around the arcade to the equator in all quadrants to detect ROP / immature / mature retina.

Once treatable stage of ROP was detected parents were contacted and with consent treatment was done. Babies which were having fully vascularized retina were not called for follow up examinations. Babies who were having immature retina or having any stage ROP were called for follow up examination depending on the severity of ROP. Babies who were having stage 3 ROP in zone 1, stage 2 or 3 ROP in zone 2 with plus disease were advised treatment.

LOCATION OF DISEASE

To define the anterior and posterior extent of ROP, the eye is divided into three zones, zone I being the most posterior and zone III being the most anterior. Zone I include all retina within a circle having a radius double the distance between the optic nerve and fovea and centered on the nerve

From zone I anterior boundary to the nasal Ora serrata, there is zone II. Zone III is the remaining temporal crescent. The zones vascularized sequentially; hence, more posterior disease occurs with more avascular retina and a greater likelihood for severe disease.[37]

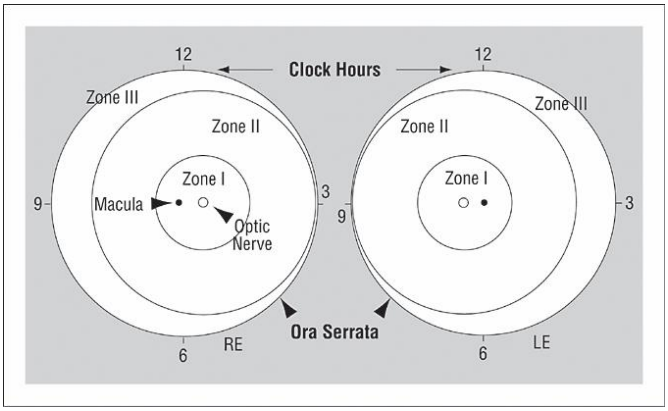


Figure 1: Retina of right eye (RE) and left eye (LE) showing zone borders and clock hours describing the location and extent of retinopathy of prematurity[37]

EXTENT OF DISEASE

The 3-o'clock position is to the right and nasal in the right eye and temporal in the left eye, while the 9-o'clock position is to the left and temporal in the right eye and nasal in the left eye. The extent of the illness is measured in hours of the clock or as 30° sector.[38] (Figure 1).

SEVERITY: STAGING THE DISEASE

The retinal vascularization is inadequate or "immature" in the preterm newborn before ROP develops. (Figure 2) The aberrant vascular response at the intersection of the vascularized and avascular retina may be broken down into five stages. The staging for the eye is determined by the most severe symptom present, even when more than one ROP stage may be present in the same eye. Although, for purpose of recording the complete examination, every stage is defined and the extent of every stage by clock hours or sector is documented.

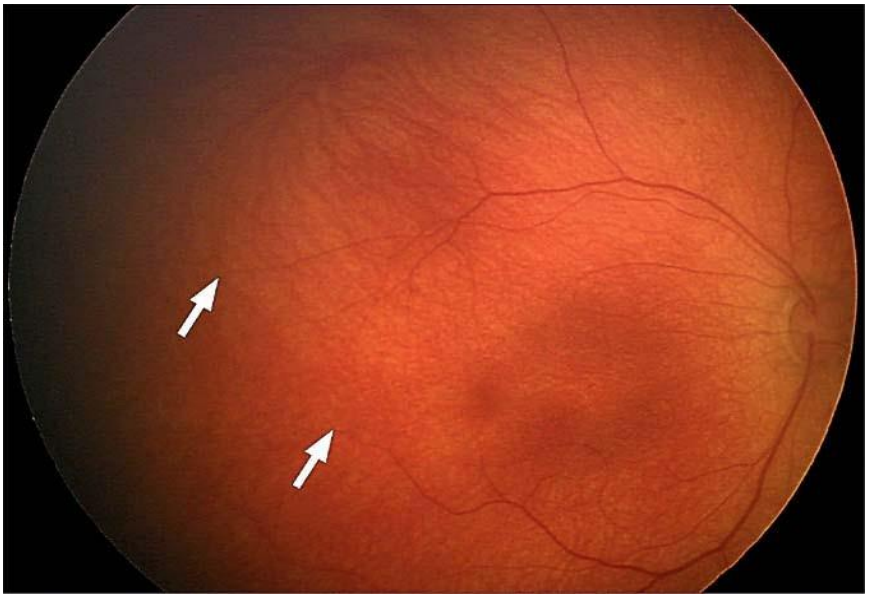


Figure 2: Fundus photograph demonstrating immature retinal vascularization in the right eye. Arrows show the progressive tapering of retinal vessels.[38]

Stage 1: Demarcation Line

Stage 1 denotes a visible line of demarcation between the posterior vascularized retina and the anterior avascular retina. Almost all children who develop stage 1 ROP will develop normally vascularized retina and vision. The demarcation line, which is generally flat, white, and located within the plane of the retina, has aberrant branching of the arteries that lead up to it. (Figure 3).

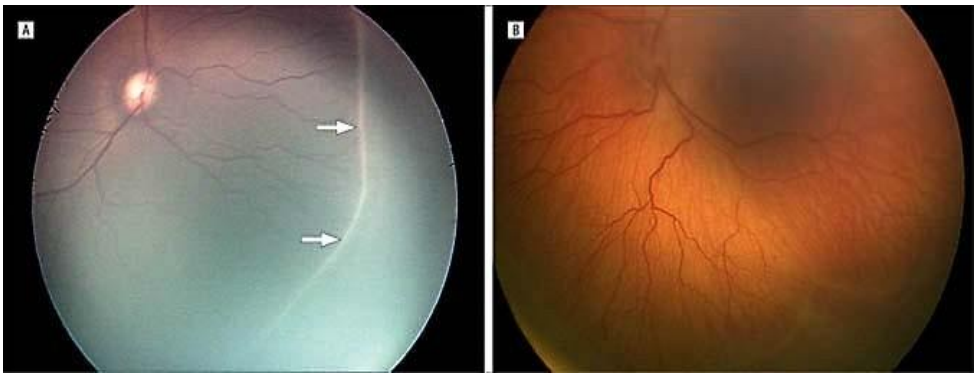


Figure 3: Fundus showing demarcation line of stage 1. A demarcation line in stage 1 with ROP.B. without ROP.[38]

Stage 2: Ridge

Stage 2 ROP is characterized by a ridge that appears around the demarcation line and has a height and breadth that ascend above the retinal plane, involves pathologic neovascularization; moreover, this neovascular response is confined within the retina. The focal growth of blood vessels within the retina increases retinal thickness and on ophthalmoscopy, it appears as a ridge at the vascular/avascular junction. (Figure 4)

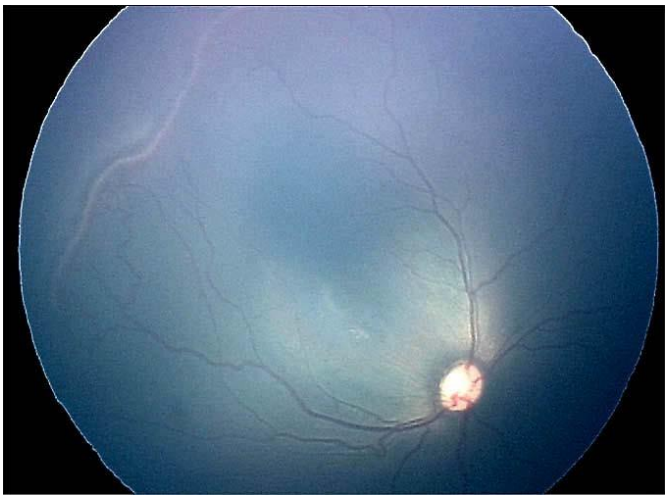


Figure 4: Pigmented Fundus photograph showing stage 2 retinopathy of prematurity at the junction between vascularized and avascular retina.[38]

Stage 3: Extra retinal Fibro-vascular Proliferation

In this stage, extra retinal fibrovascular proliferation or ridge's neovascularization spreads across the vitreous. (Figure 5) As the proliferation grows more widespread, the additional retinal growing tissue extends along the posterior portion of the ridge, giving the area a ragged look. Depending on the amount of extra retinal fibrovascular tissue that invades the vitreous, a stage 3 lesion can be classified as mild, moderate, or severe.

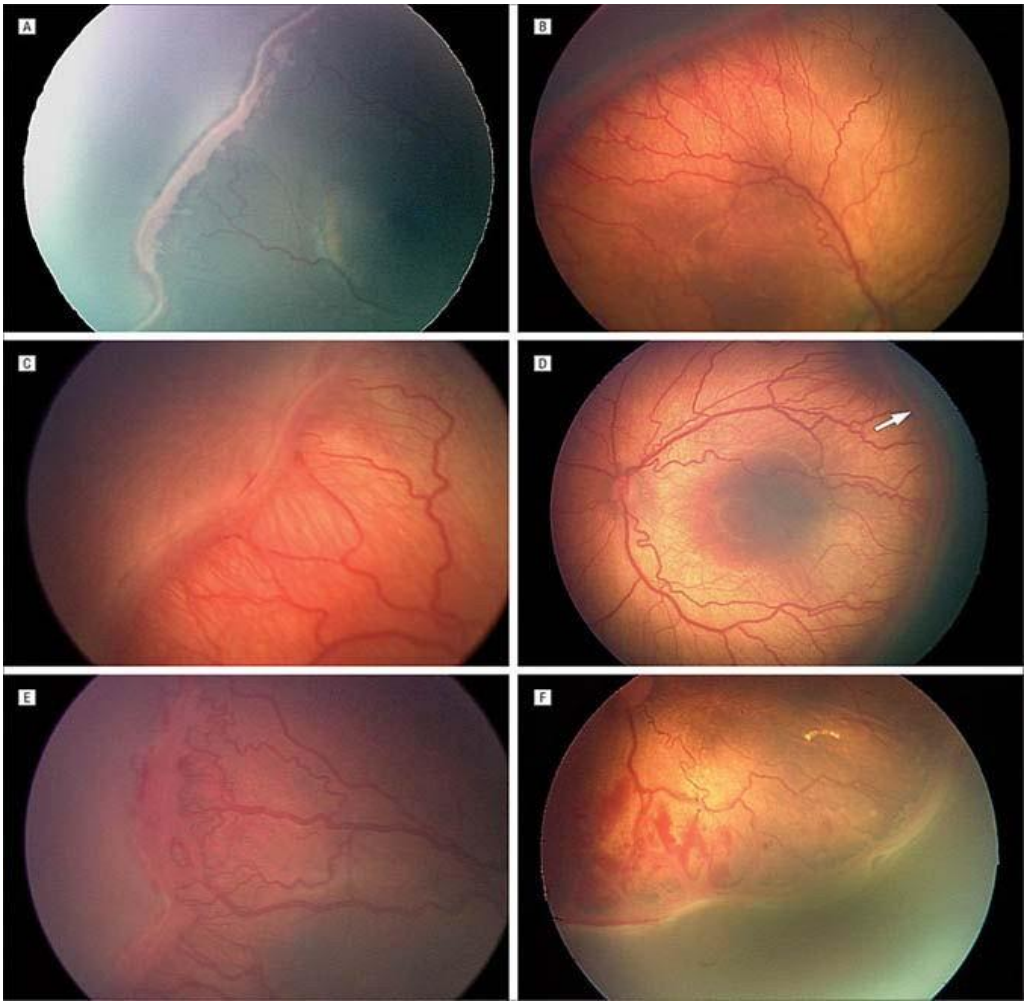


Figure 5. Fundus photographs of mild to severe stage 3 retinopathy of prematurity (ROP). A, View of mild stage 3 ROP. B, View of stage 3 moderate ROP with finger like extensions posterior to the ridge. C, View of moderate stage 3 ROP. D, View of moderate stage 3 ROP. Substantial amounts of extra retinal fibrovascular proliferation are seen infiltrating the vitreous posterior to the ridge. E, View of severe stage 3 ROP with massive infiltration of neovascular tissue surrounding the ridge. F, View of severe stage 3 ROP with infiltration of the vitreous with a dominantly fibrotic proliferation.[39]

Stage 4: Partial Retinal Detachment

Partial Retinal Detachment at Stage 4 It is split into extra foveal (stage 4A) and foveal (stage 4B) partial retinal detachment. (Figure 6) The length of fibrovascular traction and the amount of their contraction determine the degree of retinal detachment. The site of fibrovascular connection to the vascularized retina is where retinal detachment often begins.

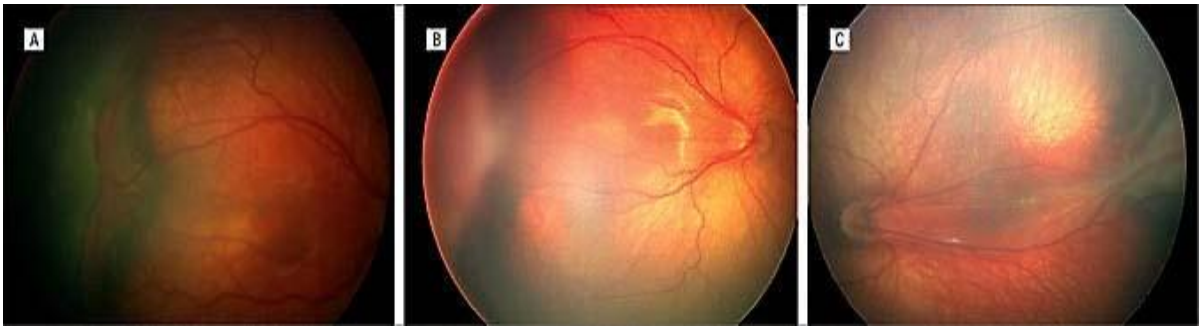


Figure 6: Stages 4A and B ROP. A→ stage 4A, extra foveal partial retinal detachment. B→ Fundus photograph of stage 4B, partial retinal detachment involving macula. C→ Fundus photograph showing stage 4B retinal detachment with extensive temporal dragging of vessels and macula.[39]

Stage 5: Total Retinal Detachment

Retinal detachment is mostly tractional and usually has a concave configuration and extends to the optic disc. (Figure 7)

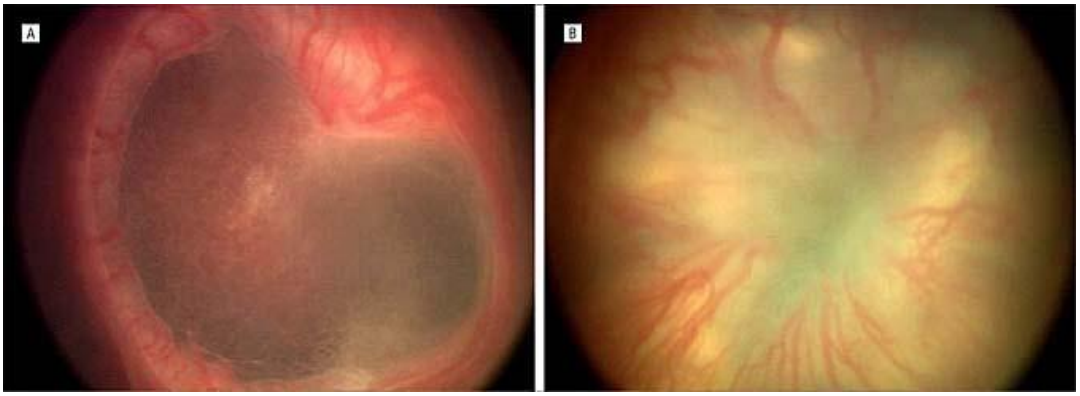


Figure 7: Fundus views of stage 5 ROP. A→ View of stage 5 ROP, total retinal detachment with open funnel configuration. B→ View of stage 5 funnel retinal detachment that is open anteriorly but narrowed posteriorly.

PRE-PLUS DISEASE

Pre-plus disease is described as vascular anomalies of the posterior pole that show more arterial tortuosity and venous dilatation than normal but are not adequate for the diagnosis of plus disease. With time, the vessel abnormalities of pre-plus may progress to frank plus disease as the vessels dilate and becomes more tortuous. (Figure 8).

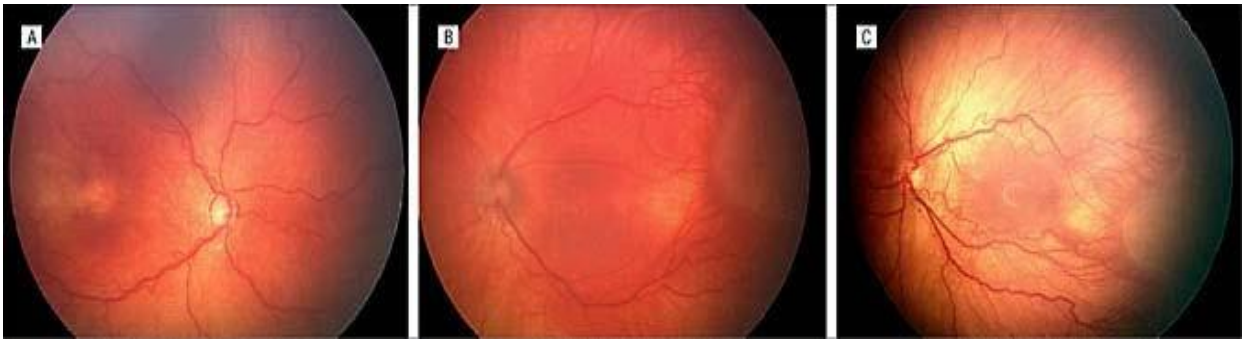


Figure 8: Examples of pre-plus disease. A, B & C Pre-plus disease with greater tortuosity of posterior pole vessels than normal but insufficient to be designated plus disease.[38]

PLUS DISEASE

As neovascularization advances, the hemodynamic conditions within the previously established retinal vessels changes. These involves increased iris vascular engorgement, inadequate pupillary dilatation (rigid pupil), and vitreous haze all worsen the condition of the posterior retinal arteries' venous dilatation and arteriolar tortuosity. Plus disease is denoted in terms of quadrants of involvement, not as clock hours and diagnosis is made if there is sufficient vascular dilatation and tortuosity present in at least 2 quadrants. (Figure 9).

Figure 9: Fundus photograph of posterior venous dilatation and arteriolar tortuosity characteristic of plus disease.[38]

AGGRESSIVE POSTERIOR ROP

Aggressive posterior ROP is rapidly progressive, severe form of ROP, posterior in location, in addition to illness and the retinopathy's ill-defined characteristics. It often progresses to stage 5 ROP if neglected. In contrast to the peripheral retinopathy, the posterior pole vessels exhibit significant dilation and tortuosity in all 4 quadrants. These vascular modifications progress quickly and difficult to differentiate between arterioles and venules because of the significant dilation and tortuosity of both vessel types. This rapidly progressing retinopathy was previously known as Rush disease. (Figure 10).

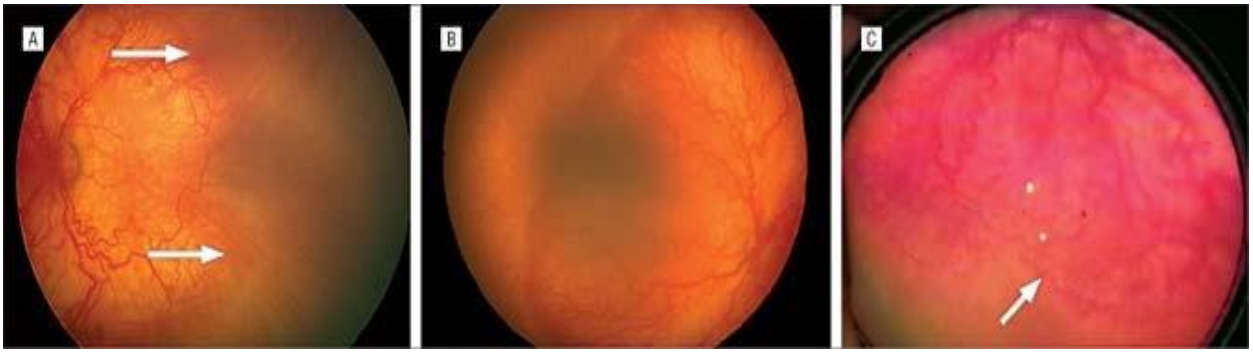


Figure 10: A, View of AP-ROP. Showing posterior location, prominence of plus disease, and modest-appearing proliferative retinopathy (arrows). B, Fundus photograph of AP-ROP demonstrating plus disease associated with brush like neovascular proliferation at junction between vascular and avascular retina. C, circumferential vessel at edge of vascularized retina.[38]

Indian guidelines for ROP screening:

Whom to screen?

There is an excessive increase in the incidence of retinopathy of prematurity (ROP) in the developing countries including India, which is referred as the third epidemic of ROP. The most important constituent of any ROP management program is planned screening strategy. ROP is seen in larger, bigger birth weight babies in Asia and other developing countries.

1. Born <34 weeks gestation
2. Gestation not known at birth but birth weight <2000 gms
3. Born at 34-36 weeks gestation AND any 1 of the following
    - Respiratory support requirement
    - Oxygen therapy for more than 6 hours
    - Sepsis
    - Apnea
    - Requires blood transfusion/exchange transfusion/unstable clinical course as determined by the pediatrician.

When to screen?

The American academy of pediatrics (AAP) recommends screening of all eligible babies at four to six weeks chronologic age (CA) or 31-33 weeks postconceptional age (PCA) whichever is later.[40] ROP can be detected prior to reaching threshold stage if first examination is done before 32 weeks PCA, which is the earliest age for significant ROP to occur.[41] Time of examination for ROP depends upon the window period. In India ROP is mostly seen in babies with low birth weight & less gestational age, so earlier examinations are essential.

What is the 'screening window'?

Progression of ROP follows a separate scenario according to the post-menstrual age of the baby. Rarely any ROP is detected before 32 weeks of gestation. The median age for detection of stage 1 ROP is 34 weeks. Pre-threshold ROP visible at 36 weeks of post-menstrual age and threshold ROP at 37 weeks. Vascularization gets completed by 40 weeks of gestation. So, the crucial period for detection of ROP is from 32 to 40 weeks of post-menstrual period. The integral phase is from 34-35 weeks to 37-38 weeks age during which the progression of the disease takes place and treatment have to be started. It may also be noted that ROP generally does not develop before 2 weeks of postnatal age.[42]

SCREENING TIME FOR THE INFANTS AT RISK

Screen all eligible babies <1.7 kg.

31 weeks PCA or 3-4 weeks after birth, whatever is earlier.

Infants less than 1.2 kg at birth and those born at 24 -30 weeks gestational age are screened early, generally not more than 2-3 weeks after birth.

No examination is required in first 2-3 weeks after birth.

One screening session must be completed before day 30 of life.

Risk factors for ROP development should be determined.

Follow up will be done at least for 3 months.

FIGURE 11: EYE SPECULUM INSERTION

FIGURE 12: EXAMINATION WITH INDIRECT OPHTHALMOSCOPE

STATISTICAL ANALYSIS

- The data will be collected and entered in MS excel (latest version). All the data will be represented in the form of number and percentages.
- Graphical representation of the variable will be shown to understand the results clearly and to measure the association for categorical dataset will be analyzed using Appropriate Test.

RESULT

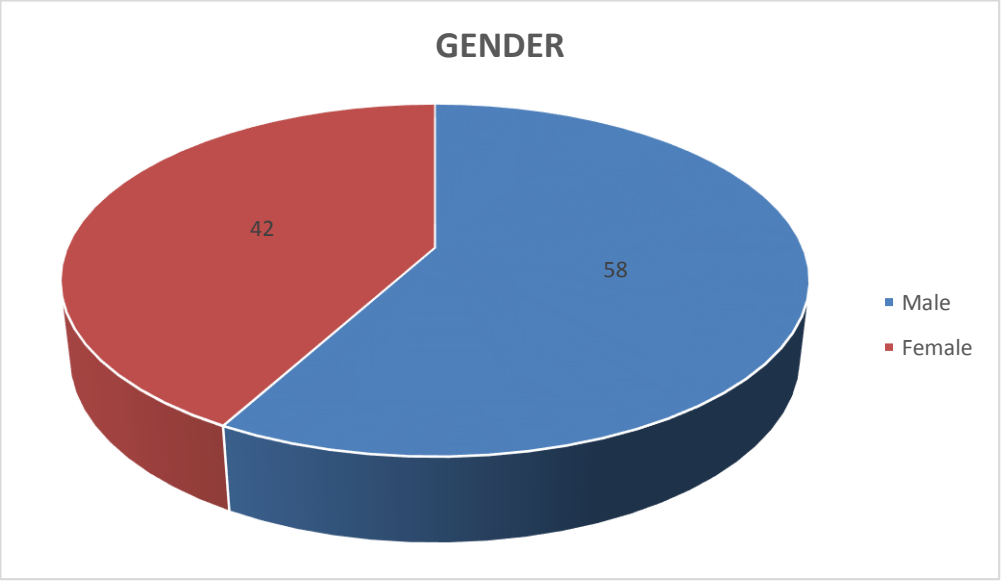
The study was conducted from August 2020- August 2021. During the study period a total of 50 babies were screened for ROP and 04 (8%) of them turned out to be positive. Our results were analyzed against the following variables Gestational age, birth weight, oxygen therapy, anemia, ventilation apnea, blood transfusion.

GENDER DISTRIBUTION:

TABLE 1: GENDER DISTRIBUTION

GENDER	NO. OF BABIES	%
MALE	29	58
FEMALE	21	42
TOTAL	50	100

GRAPH 1: GENDER DISTRIBUTION



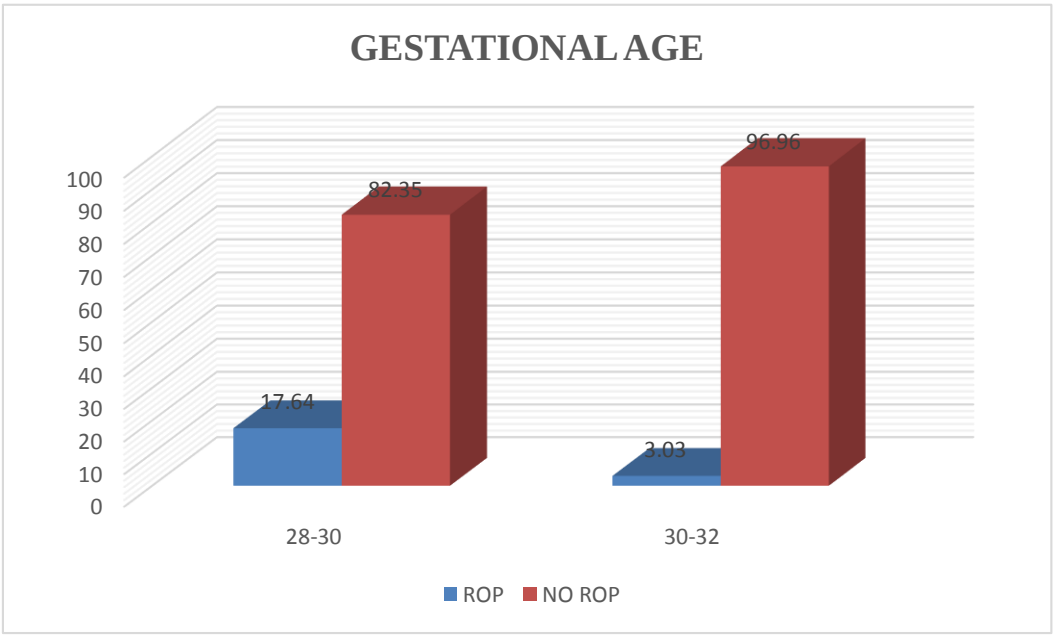
It is observed from the present study that the majority 29 (58%) babies were males, remaining 21 (42%) babies were females, among the total of 50 babies recruited in the study.

ROP AND GESTATIONAL AGE:

TABLE 2. GESTATIONAL AGE AND ROP

GESTATIONAL AGE (WEEK)	POSITIVE CASES	%	NEGATIVE CASE	%	TOTAL CASES	%
28-30	3	17.64	14	82.35	17	34
30-32	1	3.03	32	96.96	33	66
TOTAL	4	8	46	92	50	100

GRAPH 2. GESTATIONAL AGE AND ROP



In our study majority of cases of ROP occurred between 28-30 weeks (n=03,17.63%) of gestation. In 30-32 weeks of gestation 1(3.03%) case of ROP was seen. As the gestational age decreased the prevalence of ROP increased.

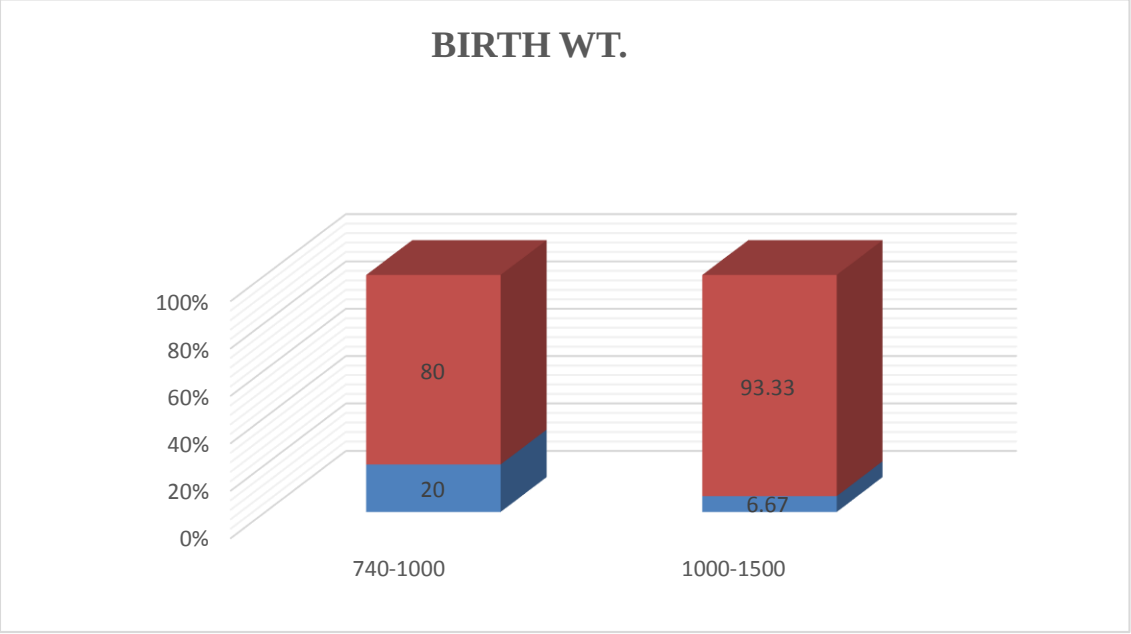
BIRTH WEIGHT AND ROP:

In our study the birth weight ranged from 740 gm to 1500 gm. Table 2 and Fig 2 shows the birth weight distribution.

TABLE 3: BIRTH WEIGHT AND ROP

	POSITIVE		NEGATIVE		TOTAL	
BIRTH WT.(gm)	NO.	%	NO.	%	NO.	%
740-1000	1	20	4	80	5	10
1000-1500	3	6.67	42	93.33	45	90
TOTAL	4	8	46	92	50	100

GRAPH 3: BIRTH WEIGHT AND ROP



20% of cases between 740-1000 gm (extremely low birth weight) birth weight developed ROP. 6.67% of babies between 1000-1500 gm birth weight developed ROP.

SEX DISTRIBUTION AND ROP:

In a total of 50 babies, there were 29 males and 21 females.

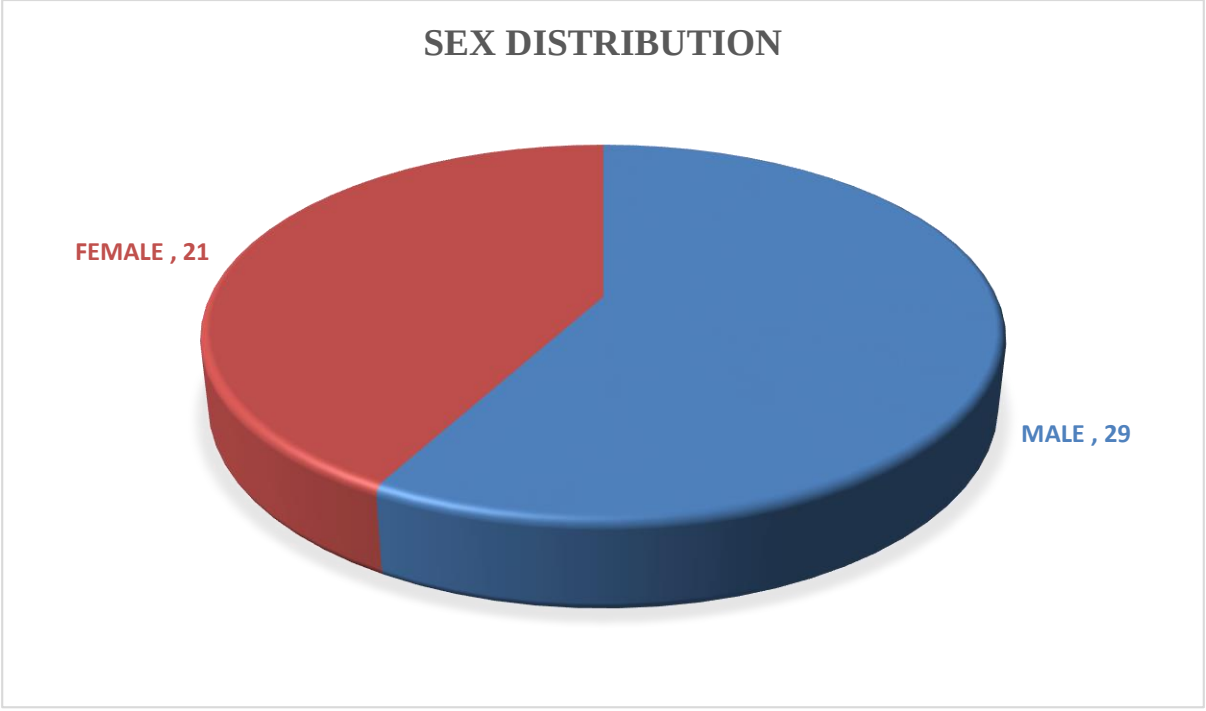
In 04 ROP positive babies there were 2 male babies and 2 female babies.

The following pie diagram and table shows the distribution.

TABLE 4: SEX DISTRIBUTION AND ROP

GENDER	POSITIVE		NEGATIVE		TOTAL	
	NO.	%	NO.	%	NO.	%
MALE	2	6.89	27	93.1	29	58
FEMALE	2	9.52	19	90.47	21	42
TOTAL	4	8	46	92	50	100

GRAPH 4: SEX DISTRIBUTION IN ROP POSITIVE CASES

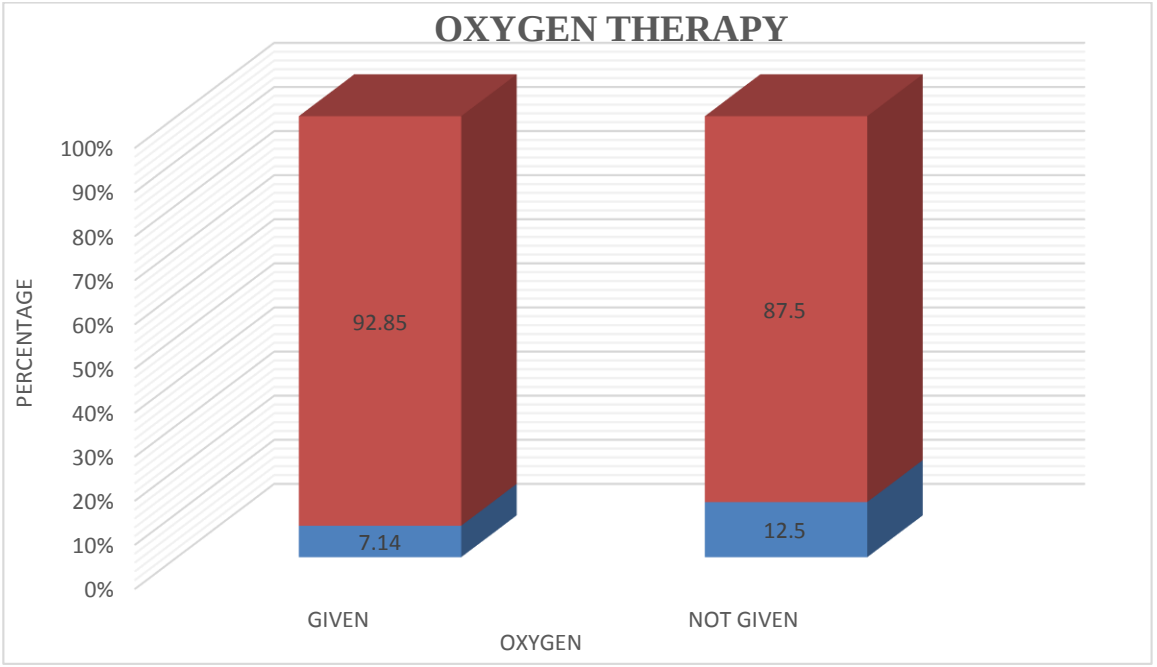


OXYGEN THERAPY AND ROP:

TABLE 5: OXYGEN THERAPY AND ROP OUTCOME

OXYGEN	POSITIVE		NEGATIVE		TOTAL	
	NO.	%	NO.	%	NO.	%
GIVEN	3	7.14	39	92.85	42	84
NOT GIVEN	1	12.5	7	87.5	8	16
TOTAL	4	8	46	92	50	100

GRAPH 5: OXYGEN THERAPY AND ROP OUTCOME



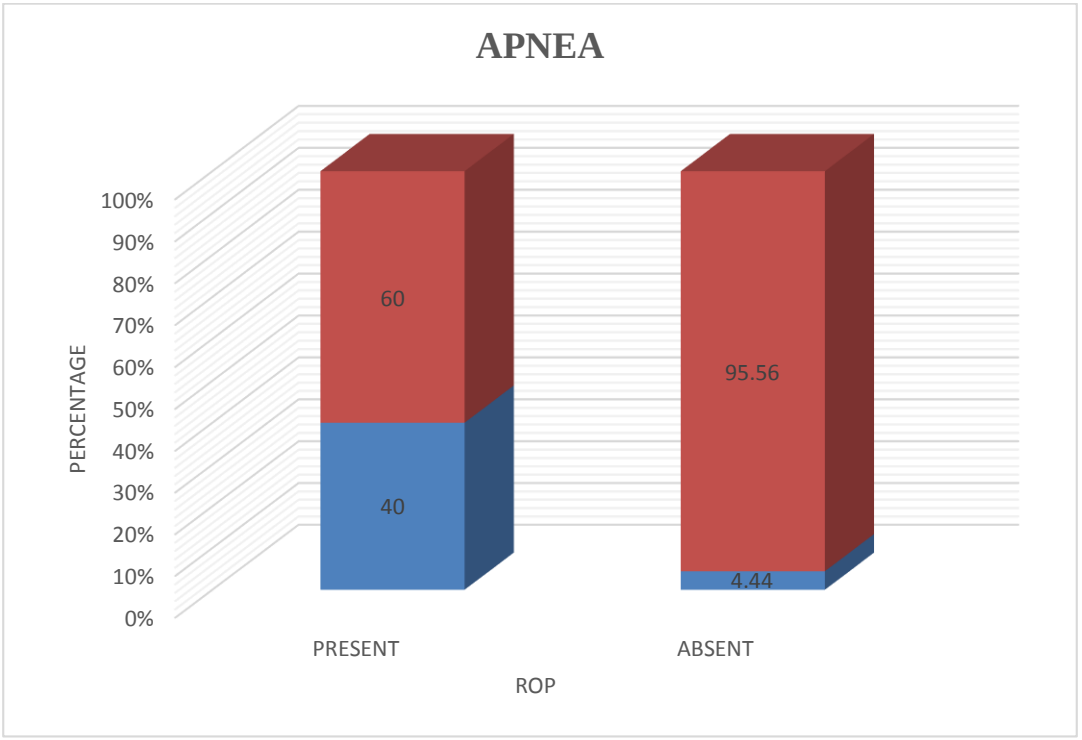
In our study out of 42 babies who received oxygen, 7.14% (n=3) of babies developed ROP. 12.5% (n=1) babies who have not received oxygen developed ROP.

APNEA AND ROP:

TABLE 6: APNEA AND ROP

APNEA	POSITIVE		NEGATIVE		TOTAL	
	NO.	%	NO.	%	NO.	%
PRESENT	2	40	3	60	5	10
ABSENT	2	4.44	43	95.56	45	90
TOTAL	4	8	46	92	50	100

GRAPH 6: APNEA AND ROP



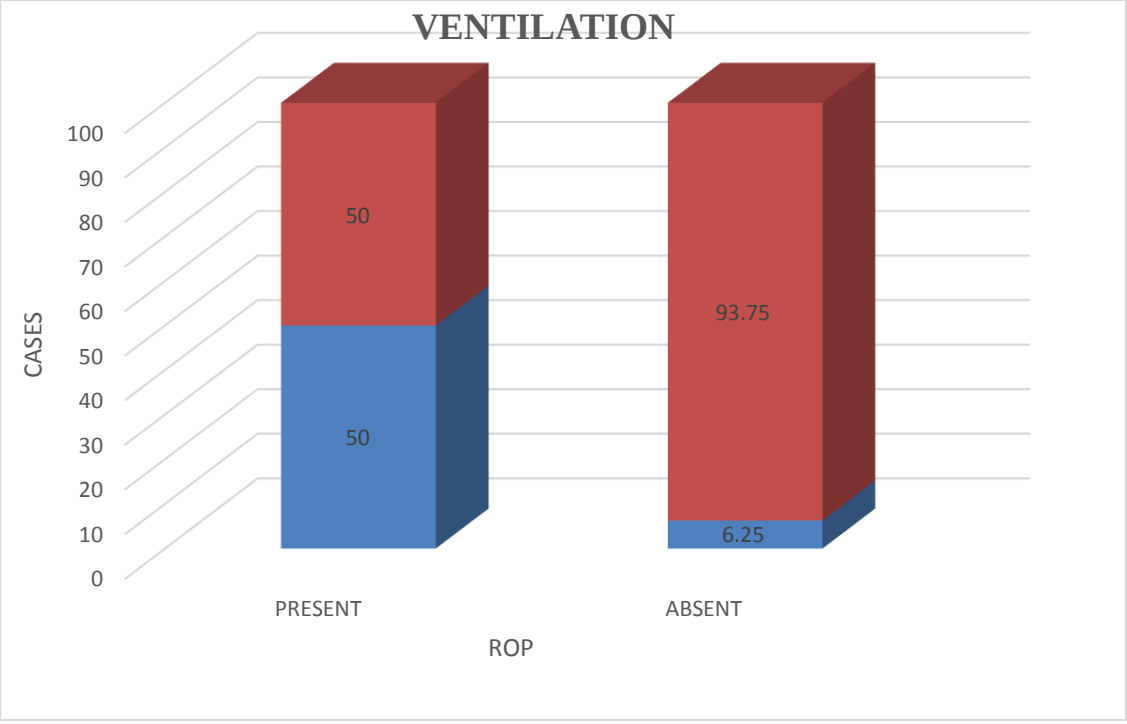
Out of 50 cases 5 cases (10%) had apnea. Out of 13 cases 10 cases(76.9%) had ROP. In 04 ROP positive cases 2 cases (50%) cases had apnea.

VENTILATION AND ROP:

TABLE 7: VENTILATION AND ROP

VENTILATION	POSITIVE		NEGATIVE		TOTAL	
	NO.	%	NO.	%	NO.	%
PRESENT	1	50	1	50	2	4
ABSENT	3	6.25	45	93.75	48	96
TOTAL	4	8	46	92	50	100

GRAPH 7: VENTILATION AND ROP



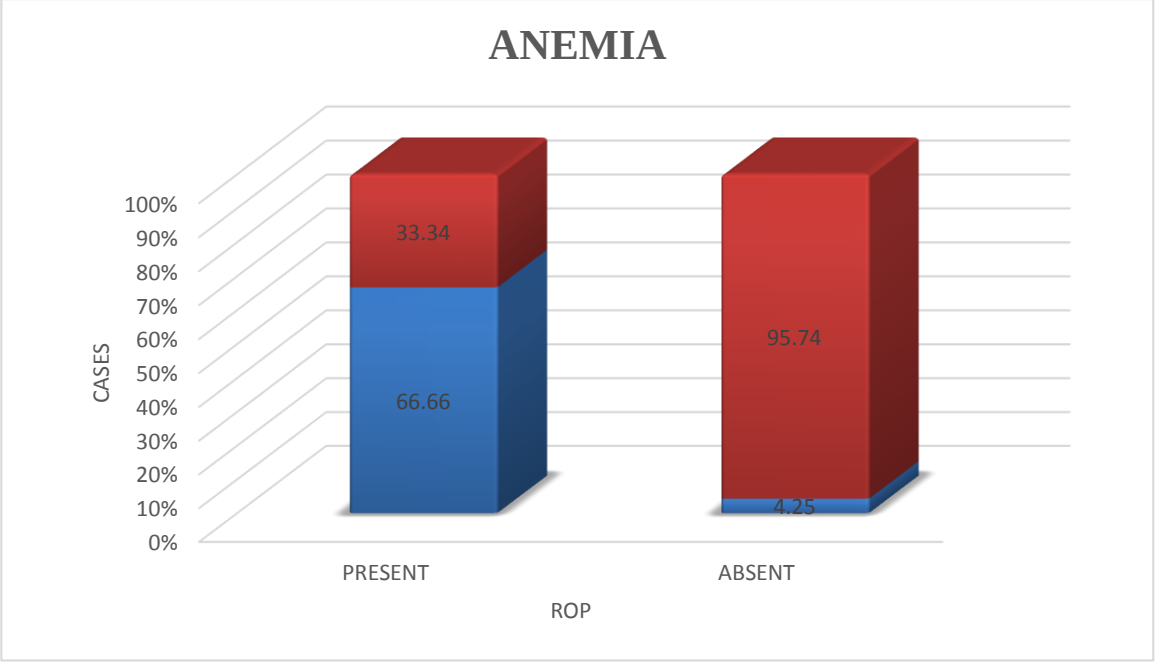
Out of 50 babies 2 babies (4%) received ventilation in which 1 case (50%) had ROP. Out of 04 ROP positive cases 1 cases (25%) received ventilation.

ANEMIA AND ROP:

TABLE 8: ANEMIA AND ROP

ANEMIA	POSITIVE		NEGATIVE		TOTAL	
	NO.	%	NO.	%	NO.	%
PRESENT	2	66.66	1	33.34	3	6
ABSENT	2	4.25	45	95.74	47	94
TOTAL	4	8	46	92	50	100

GRAPH 8: ANEMIA AND ROP



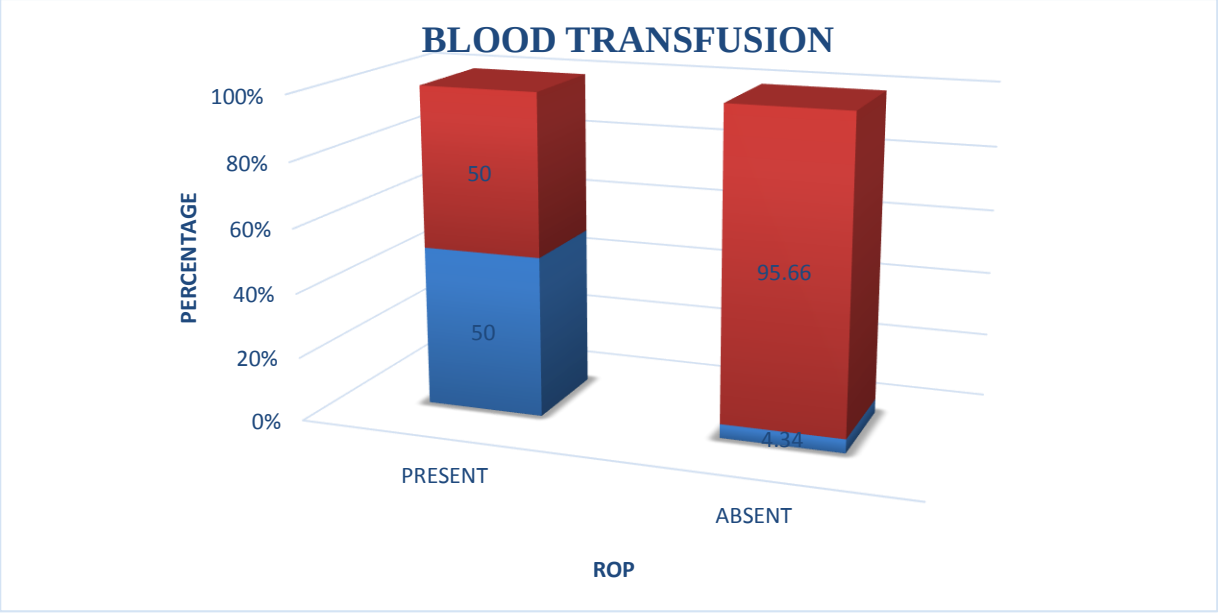
Out of 50 babies 03 babies (6%) had anemia. Out of 03 babies 2 (66.66%) developed ROP. Out of 04 ROP positive babies 2 (50%) were found to be anemic.

BLOOD AND BLOOD PRODUCT TRANSFUSION AND ROP:

TABLE 9: BLOOD AND BLOOD PRODUCT TRANSFUSION AND ROP

BLOOD TRANSFUSION	POSITIVE		NEGATIVE		TOTAL	
	NO.	%	NO.	%	NO.	%
PRESENT	2	50	2	50	4	8
ABSENT	2	4.34	44	95.66	46	92
TOTAL	4	8	46	92	50	100

GRAPH 9: BLOOD & BLOOD PRODUCT TRANSFUSION AND ROP



Out of 50 babies 04 babies (8%) received blood and blood product transfusion. Out of 4 babies 2 babies (50%) developed ROP. Out of 04 ROP positive cases 2 babies (50%) developed ROP.

TABLE 10: FREQUENCY DISTRIBUTION OF STAGES OF ROP

STAGES	NO OF CASES
1	1(25%)
2	2(50%)
3	1(25%)
4	NIL
5	NIL
APROP	NIL
TOTAL CASES	4

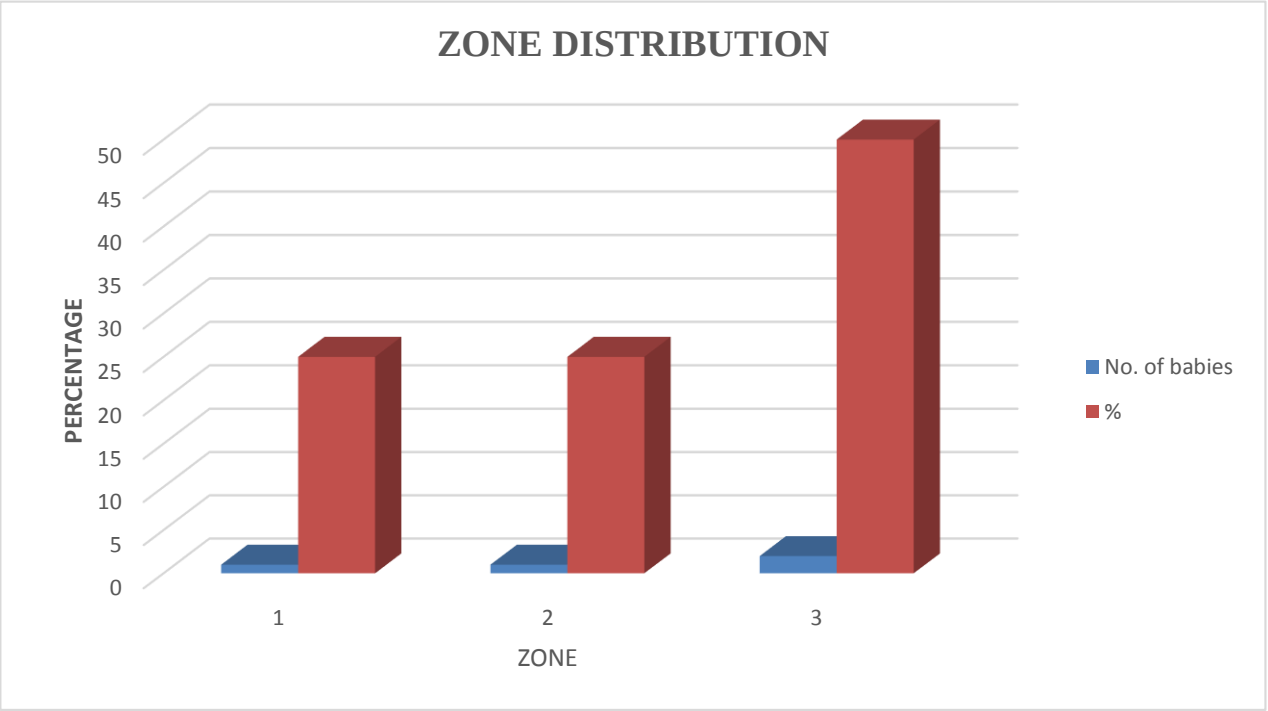
Out of 04 positive cases 25% cases are in stage 1, 50% are in stage 2, 25% are in Stage 3, none of the cases were found in stage 4, stage 5 & APROP.

TABLE 11: ZONE DISTRIBUTION OF ROP

In the present study ROP distribution was seen to be equal in Zone 1&2 representing 1 baby each (25%). However, Zone 3 ROP, was seen in 2 babies (50%).

ZONE	NO. OF BABIES	%
1	1	25
2	1	25
3	2	50
Total	4	100

GRAPH 11: ZONE DISTRIBUTION OF ROP



DISCUSSION

ROP AND GESTATIONAL AGE

In our study we have enrolled 50 babies with birth weight <1500 gm and gestational age <32 weeks. We also studied the risk factors associated with this disease seen in these babies. In our study, the prevalence of retinopathy of prematurity was 04 (8%). This was in concurrence with study done by Maheswari et al. in which all babies weighing <1500 gm and a gestational age <35 weeks were screened.[43] Rekha S et al screened all babies with birth weight <1500 gm or and gestational age >35weeks.[44] In a recent article, Kaul S et al. (2021) have suggested the same screening criteria, where prevalence was 28%.[45] Chaudhari S et al.[46] screened all babies with birth weight <1500 gm and gestation >32 weeks. Infants with birth weight >1500 gm and gestation more than 32 weeks were screened only if they had additional risk factors. Sharma P et al.[47] screened all babies with birth weight <1500g and gestation <32 weeks, Infants with birth weight 1501-1800 gms or 33-34 weeks if they had additional risk factors. In a study done by Anudeep et al., out of the 65 preterm babies screened, 24 (37%) developed ROP.[48] Vinekar et al.[49] suggested that the scenario in developing countries is quite different. As compared to infants in Western nation, gestationally "older" newborns are more prone to have ROP. Hence, the application of Western screening guidelines for developing countries has been questioned by Jalali et al.[50] Goble et al.[50] felt that they were screening too many babies for ROP and recommended that babies with birth weight above 1250 g should not be screened. ROP and gestational age in our study the positive cases of ROP in babies with gestational age ≤32 weeks is 8%. In that 28-30 week is 17.64% 30-32 weeks is 3.03% prevalence increased as the gestational age decreased.

ROP AND BIRTH WEIGHT

In our study the prevalence of ROP is 8% In ELBW (<1000 gm) babies the prevalence is 20%, in babies with birth weight 1000-1500 gm the prevalence is 6.67%. In the western studies, the prevalence of ROP has been reported to be 28% in less than 1500 gm babies. In the West ROP, at least the threshold variety is not seen in higher birth weight babies. In contrast ROP is seen in larger, bigger birth weight babies in Asia and other developing countries. In South India, threshold ROP has been seen in babies born with 2000 g birth weight. While partly this might reflect the failure of very small infants to thrive, other factors such as perhaps the quality of neonatal care that has led to a decline of ROP in the West is lacking here. Of note, a similar scenario existed in Lithuania, where in ROP was seen in larger infants initially.

However, the birth weights of babies with ROP have fallen quickly due to improvements in neonatal care. A similar swing in the pendulum could be expected to occur in India as well! Nevertheless, it is essential to realize that at least in the present scenario, the cut off birth weight and the gestational ages of our babies that need to be screened for ROP need to be higher. Prematurity is the single most important risk factor responsible for retinopathy of prematurity. As reported by Palmer, et al.[50] lower birth weight and younger postconceptional age were strongly associated with ROP incidence and severity. In a study done by Chaudhari S et al the Incidence was 83% in 28-30 weeks, 32% in 31-33 weeks.[50]

ROP AND OXYGEN THERAPY

In our study prolonged oxygen therapy was found to be a significant risk factor. We found that mean duration of oxygen was significantly higher in the ROP group. This was similarly seen in study done by Abdel et al.[51] in which 172 infants were screened and there was positive association between ROP and oxygen supplementation. Study done by Rekha S et al.[44] also have shown a similar significance with the duration of oxygen therapy. In the study done by Ratra et al.[52] there was significant association between ROP and oxygen supplementation (p value = 0.001). In a study by Ved Gupta et al.[53] in their center, Oxygen (p=0.01) was an independent risk factor. In a study done by Chaudhari S et al.[50] Gupta VP et al.[54] oxygen was found to be significantly associated with ROP. Study done by Dutta S et al.[55] has concluded that there was no significant association of ROP with oxygen therapy.

ROP AND APNEA

In our study apnea found to be significant risk factor. In a study done by Agarwal R et al.[56] in 2002 apnea came as a significant risk factor. In another study by Gupta et al.[51] in 2004 apnea came as a significant risk factor. In another study by Chaudhary S et al.[50] in 2009 apnea came as a significant risk factor.

ROP AND VENTILATION

In our study ventilation found to be highly significant risk factor. In a study done in Iran by Mokhtari MB et al mechanical ventilation came as a significant risk factor.[57] In a retrospective study done in Singapore by Shah VA et al has shown that ventilation as a significant risk factor.[58] ROP and ANEMIA in our study anemia found to be significant risk factor.

In another study by Rekha S et al.[44] in 1996 anemia came as a significant risk factor. In a study done by Li Liu et al.[59] from China, anemia was a significant risk factor.

ROP AND BLOOD TRANSFUSION

In our study there is a highly significant association between blood and blood products transfusion. In a study done in Brazil by Pinheiro AM et al.[60] there was a significant association between blood transfusion and ROP. In a study done in Egypt by Abdel H.A.A. hakeem et al.[51] there was a significant association between blood transfusion and ROP. In a study done in Iran by Mojginbayat-Mokhtari et al. [57] there was a significant association between blood transfusion and ROP.

ROP AND STAGES

Maximum number of babies i.e., 2 (50%) had stage 2 ROP, 1 (25%) baby were having stage I ROP and stage III ROP respectively. No babies had stage 4, 5 APROP. Our finding was similar to a study done by Anudeep et al, 30% had stage 1, 37% had stage 2, and 33% had stage 3.[48]

ROP AND ZONE

Maximum number of babies i.e., 2 (50%) had ROP in zone 3, 2 (25%) babies had ROP in zone 1 & 2 respectively. Our finding was similar to a study done by Crystal Le in which ROP was most commonly seen in Zone III (68%) and Zone II was the second most common (26%).[61]

CONCLUSION

ROP is a disorder of developing retinal blood vessels in the premature infant retina.
ROP is the commonest and more preventable form of blindness. During the study period of 1 year from August 2020 to August 2021 babies were screened for ROP in which 04 were found to be positive.

Our PREVELENCE rate is 8%. ROP is found to be associated with the following risk factors in our study Oxygen therapy, blood transfusion, ventilation and anemia. Out of 04 positive babies for ROP 01 baby was <1000 gm and 03 babies were >1000 gm.

SUMMARY

The present study of 50 infants who were screened for ROP to know "PREVALENCE OF ROP IN PREMATURE INFANTS" Out of 50 babies 29 were males and 21 were females. Out of 50 babies 04 babies were found to be positive for ROP. All babies with gestational age <32 weeks or birth weight <1500gms. Out of 04 positive ROP cases 02 males and 02 females were present. Prevalence of ROP was equal in both sexes. As the gestational age is decreased the prevalence of ROP increased. prevalence of ROP between 28-30 weeks is 17.64%,30-32 weeks 3.03%. Out of 04 ROP cases 1 was below 1000 gm, 03 were above 1000gms. Out of 42 babies who received OXYGEN,07.14%[n=3] were ROP positive and it is found to be a significant risk factor in our study. Out of 03 babies with anemia, 66.66%[n=02] were positive for ROP. Anemia was found to be a significant risk factor. Out of 02 babies who received ventilation ,50%[n=1] was positive for ROP. Ventilation is found to be a significant risk factor. Out of 05 babies with apnea ,40%[n=2] were positive for ROP.
Apnea is found to be a significant factor for ROP in our study. Out of 04 babies with ROP 1 babies (25%) had stage 1 ROP,2(50%) had stage 2 ROP,1(25%) had stage 3 ROP, no babies had APROP. No babies were found in stage 4 and 5.

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(ANNEXURE – 1: PROFORMA)

SR. NO. **Date:**

Registration no:

Name:

Age/Sex:

DOB:

Address:

Ph. No.

Gestational age:

Birth order:

Post natal age:

Risk factors:

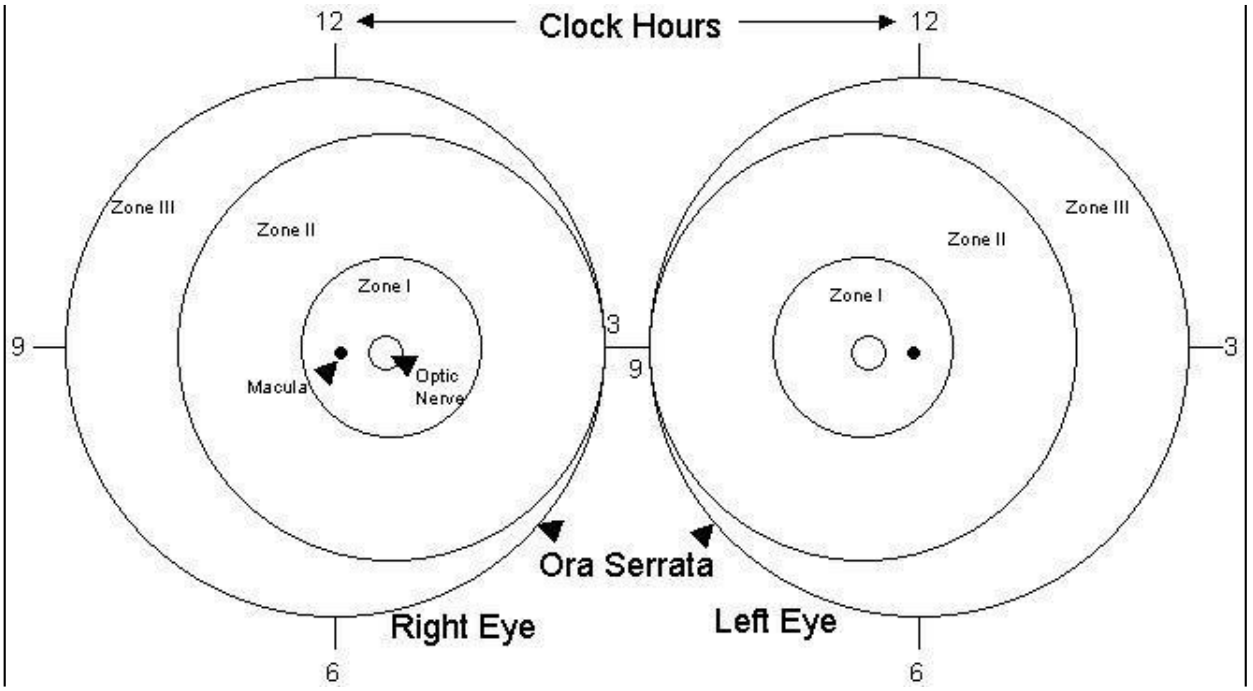
Associated systemic conditions:

EXAMINATION: **RE** **LE**

A/S:

PUPIL:

FUNDUS:



IMMATURE/MATURE
ZONE:
STAGE OF ROP:
CLOCK HOURS INVOLVED:
PLUS DISEASE: YES/NO
REGRESSING ROP STAGE:

IMMATURE/MATURE
ZONE:
STAGE OF ROP:
CLOCK HOURS INVOLVED:
PLUS DISEASE: YES/NO
REGRESSING ROP STAGE:

IMPRESSION:

TREATMENT:

(ANNEXURE 2: INFORMED CONSENT)

Santosh Medical College and Hospital Informed Consent

I Mr/Mrs
Resident
of.....

Hereby declare that I give my full and free & voluntary informed consent in the thesis entitled **"PREVALENCE OF RETINOPATHY OF PREMATURITY IN PREMATURE INFANTS"**

Dr. SUKRITI GUPTA has informed to me to my full satisfaction, in the language I understand about the purpose and nature of the study & various physical examinations by the study to be carried out. I have been informed about the duration of the study and instructions to be followed during the study period.

I give full consent to enroll my baby in the above study and I reserve my rights to withdraw from the study whenever I wish prejudice of my right to undergo further treatment at Santosh Hospital Ghaziabad U.P.

Signature of Attending Signature of Parent

Name

संतोष मेडिकल कॉलेज एवं अस्पताल (गाजियाबाद)

<u>सूचितसहमति</u>

मैं _____ बेटा_____

निवासी_____ घोषणा करता हूं कि मैं डॉ._____, ने मुझे मेरी भाषा में अपने अध्ययन, अध्ययन के उद्देश्य के बारे मे, और अध्ययन के दौरान अपना ईजानेवाली विभिन्न प्रयोगशाला जांच के बारे में सूचित कर दिया है। मुझे अध्ययन की अवधि और उसके दौरान अपना एजाने वाले निर्देश भी मेरी भाषा मे समझा दिए गए हैं| मैं उपरोक्त अध्ययन में बिना किसी दबाव के दाखिला ले रहा हूं और पूर्ण सहमति दे रहा हूं| सन्तोष अस्पताल, गाजियाबाद में अपने आगेके उपचार पर बिना प्रतिकूल प्रभाव डाले इस अध्ययन से और प्रयोगशाला जांच कीप्रकृति से मैं जब चाहूँ बाहर जाने के अपने अधिकार को सुरक्षित रख रहा हूं

रोगी / रोगी के रिश्तेदार के हस्ताक्षर

नाम:

दिनांक:

मेरी उपस्थिति में मरीज ने पूरी तरह से डाकटर के बताए गए अध्ययन और निर्देशों को समझने के बाद अपनी स्वतंत्र इच्छा से ऊपरके फार्म पर हस्ताक्षर कि ए हैं।

MASTERCHART

SR. NO	IP NO.	NAME	AGE (DAYS)	SEX	POG(WKs)	BIRTH WT.(GRAM)	ANEMIA	VENTILATION	APNEA	OXYGEN	ROP	STAGE	ZONE
1	17089475	B/O baby	4	F	29	1400	-	-	-	+	-	NIL	NIL
2	17092723	B/O Anchal Kumari	4	F	28	960	-	-	-	+	-	NIL	NIL
3	17100512	B/O Kusum Sharma	8	M	31	1480	-	-	-	-	-	NIL	NIL
4	17100340	B/O Nida	13	F	29	1370	-	-	-	+	-	NIL	NIL
5	17219675	B/O Priyanka, pari	18	M	28	1450	-	-	-	+	-	NIL	NIL
6	17104683	B/O Sahian	4	F	32	1480	+	-	+	+	+	1	3
7	17101401	B/O Naina	3	F	29	1370	-	-	-	+	-	NIL	NIL
8	17108108	B/O Annu	9	M	31	1470	-	-	-	+	-	NIL	NIL
9	17104683	B/O Sahin	10	F	32	1450	-	-	-	-	-	NIL	NIL
10	17103819	B/O Jyoti	4	M	28	1100	-	-	-	+	-	NIL	NIL
11	17108108	B/O Manisha	9	M	29	1340	-	-	-	+	-	NIL	NIL
12	17101401	B/O Naina	3	M	32	1480	-	-	-	+	-	NIL	NIL
13	17108165	B/O Ikra	13	M	32	1490	-	-	-	+	-	NIL	NIL
14	17109298	B/O Pooja	12	F	29	1250	-	-	-	+	-	NIL	NIL
15	17109512	B/O Anabiya	11	M	31	1310	-	-	+	+	-	NIL	NIL
16	17109884	B/O Pooja	7	M	31	1420	-	-	+	+	-	NIL	NIL
17	17110341	B/O Anmol	8	F	28	930	-	-	-	+	-	NIL	NIL
18	17110341	B/O Parika	9	M	32	1420	-	-	-	+	-	NIL	NIL
19	17111616	B/O Kiara	5	F	29	1230	-	-	-	-	-	NIL	NIL
20	17108165	B/O Anita	6	M	31	1390	-	-	-	-	-	NIL	NIL
21	17108165	B/O ritika	7	F	29	1220	-	-	-	-	-	NIL	NIL
22	17108165	B/O nargis	8	M	32	1490	-	-	-	-	-	NIL	NIL
23	17115630	B/O Priyanka	4	M	28	880	-	-	-	-	-	NIL	NIL
24	17106489	B/O sangeeeta	6	F	31	1470	-	-	-	-	-	NIL	NIL
25	17120450	B/O swati	4	M	32	1460	-	-	-	-	-	NIL	NIL

26	17106212	B/O devi	9	M	32	1420	-	-	-	-	-	NIL	NIL
27	17106297	B/O shrushti	6	F	31	1380	-	-	-	-	-	NIL	NIL
28	17106347	B/O ishita	8	F	31	1390	-	+	+	+	+	2	3
29	17106321	B/O nirmala	8	M	28	1190	-	-	-	-	-	NIL	NIL
30	17128980	B/O panchi	7	M	32	1490	-	-	-	-	-	NIL	NIL
31	17114543	B/O tara	6	M	31	1420	-	-	-	-	-	NIL	NIL
32	17129098	B/O diksha	8	F	31	1410	-	-	-	-	-	NIL	NIL
33	17117867	B/O aarohi	8	F	31	1390	-	-	-	-	-	NIL	NIL
34	17117890	B/O somya	4	M	28	1110	-	-	-	-	-	NIL	NIL
35	17119897	B/O sheeba	9	M	32	1410	+	-	-	+	+	2	1
36	17116574	B/O sneha	2	F	32	1430	-	-	-	-	-	NIL	NIL
37	17113546	B/O richa	3	F	31	1310	-	-	-	-	-	NIL	NIL
38	17118004	B/O sakshi	4	M	32	1410	-	-	-	-	-	NIL	NIL
39	17128940	B/O tiya	5	M	32	1450	-	-	-	-	-	NIL	NIL
40	17128960	B/O harshita	3	F	28	790	-	-	-	+	-	NIL	NIL
41	17128925	B/O himanshi	9	M	32	1480	-	-	-	-	-	NIL	NIL
42	17128937	B/O parul	4	M	32	1390	-	-	-	-	-	NIL	NIL
43	17126576	B/O Maya	5	F	31	1310	-	-	-	-	-	NIL	NIL
44	17124844	B/O Prachi	3	M	32	1340	+	+	-	-	-	NIL	NIL
45	17117697	B/O jaspreet	8	F	29	1270	-	-	-	-	-	NIL	NIL
46	17128739	B/O Divya	3	M	32	1410	-	-	-	+	-	NIL	NIL
47	17124282	B/O Shobha	10	M	31	1350	-	-	-	-	-	NIL	NIL
48	17124752	B/O ayushi	5	M	32	1420	-	-	-	-	-	NIL	NIL
49	17110684	B/O Sazil	8	F	28	980	-	-	-	-	+	3	2
50	17115761	B/O Radha	10	M	32	1480	+	-	+	+	-	NIL	NIL

PLAGIARISM CHECK

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INTRODUCTION

Preterm birth can impose challenges on the developing ocular system, resulting in the various visual manifestations of varied significance and pathological scope. This ocular condition can pose severe vision loss.

Retinopathy of prematurity (ROP), or retrolental fibroplasias as it was originally named, had life span as a 20th century disease. Over the previous 59 years this disease was first correlated with prematurity by Terry in 1942, a plethora of literature has emerged on retrolental fibroplasia (RLF) and retinopathy of prematurity (ROP) (1). It has been responsible for the two largest "epidemics" of blindness in neonates in modern times. These outbreaks of the disease occurred 25 years apart in the mid-1950s and late 1970s approximately (1). It has been an "oxygen epidemic" from the late 1940s through the mid-1950s.

ROP is a bilateral proliferative retinopathy, which occurs in premature infants with low birth weight (LBW) who often have been exposed to higher concentrations of oxygen & effects the developing retinal blood vessels. It emerges out as one of the leading causes of preventable blindness and accounts for up to 10% of childhood blindness in developed countries (2). Ever since 1940s, with the arrival of improved facilities and treatment, the survival rate of premature infants has been increased & recognized as an important cause of childhood visual impairment and blindness (2).

Its prevalence in moderately developed countries human development index; HDI (30-100) is as high as 60% in premature babies & in poorly developed countries, which includes India, is 3 to 13%. The incidence in highly developed countries, those with a HDI of 1-3% is 3- 13. Its incidence is higher in babies of low gestational age and LBW (3,4.) Major risk factors of ROP include hyperoxia, sepsis, blood transfusions, acidosis, antioxidant deficiency, patent ductus arteriosus, apnoea and intraventricular hemorrhage.